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Review Article

Hematological emergencies in intensive care unit: Recognition, management protocols, and multidisciplinary care

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ABSTRACT

Hematological emergencies constitute critical medical conditions that demand immediate recognition and intervention in intensive care settings. These life-threatening disorders encompass bleeding emergencies such as disseminated intravascular coagulation and trauma-induced coagulopathy, thrombotic complications including thrombotic thrombocytopenic purpura and atypical hemolytic uremic syndrome, hemolytic crises, sickle cell-related emergencies, inflammatory conditions like hemophagocytic lymphohistiocytosis, and malignancy-associated complications. The heterogeneous nature of these conditions, combined with their rapid progression potential, necessitates systematic diagnostic approaches and evidence-based management protocols. Successful outcomes depend on early identification through clinical vigilance and appropriate laboratory investigations, prompt initiation of targeted therapies, and seamless coordination among multidisciplinary care teams including intensivists, hematologists, transfusion medicine specialists, and consulting services. The complexity of managing critically ill patients with hematological emergencies requires specialized knowledge, standardized treatment protocols, and continuous quality improvement initiatives. This comprehensive review synthesizes current evidence-based approaches to recognition, diagnosis, and management of the most prevalent hematological emergencies encountered in intensive care units, emphasizing the critical importance of timely intervention and collaborative care in reducing mortality and improving patient outcomes.

Keywords: Bleeding disorders, Coagulopathy, Hematological emergencies, Hemolysis, Intensive care unit, Multidisciplinary care, Thrombocytopenia

INTRODUCTION

Hematological emergencies in the intensive care unit (ICU) represent a diverse group of conditions that can rapidly deteriorate without prompt recognition and appropriate management. These emergencies may present as primary hematological disorders requiring ICU care or develop as complications in critically ill patients with other primary conditions.[1] The management of these complex cases requires not only immediate clinical intervention but also coordinated care from multiple specialties including hematology, critical care medicine, transfusion medicine, and various consulting services.[2]

The incidence of hematological emergencies in ICU settings has increased with advances in cancer therapy, organ transplantation, and the survival of patients with complex medical conditions. These emergencies can be broadly categorized into bleeding disorders, thrombotic

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complications, hemolytic conditions, and malignancy-related hematological crises. Each category presents unique diagnostic challenges and requires specific therapeutic approaches while considering the patient's overall critical illness status. The major categories of hematological emergencies encountered in ICU settings are summarized in Table 1.

Bleeding emergencies and coagulopathies

Disseminated intravascular coagulation (DIC)

DIC represents one of the most challenging hematological emergencies in the ICU. DIC is characterized by widespread activation of the coagulation cascade, leading to both thrombotic and bleeding complications.[3] The pathophysiology involves systemic activation of coagulation with consumption of platelets and clotting factors, ultimately resulting in a bleeding tendency despite initial hypercoagulability.[3-6]

Recognition of DIC requires a high index of suspicion in patients with predisposing conditions such as sepsis, trauma, hepatic failure, malignancy, or obstetric complications. A remote history of deep vein or arterial thrombosis may also suggest DIC.[3] Clinical presentation may include bleeding from multiple sites (gums, surgical areas, and genitourinary tract) and symptoms of organ dysfunction including hematuria, oliguria, dyspnea, hemoptysis, chest pain, and altered mental status. Physical examination reveals hemorrhage, ecchymosis, hematomas, jaundice, necrosis, gangrene, purpura, petechiae, and cyanosis. Complications include acute respiratory failure and neurological deficits from bleeding or thrombosis.[3,6]

Table 1: Classification of hematological emergencies in intensive care units.

Bleeding emergencies and coagulopathies
Disseminated intravascular coagulation
Trauma induced coagulopathy
Thrombotic emergencies
Thrombotic thrombocytopenic purpura
Atypical hemolytic uremic syndrome
Hemolytic emergencies
Autoimmune hemolytic anemia
Hemolytic transfusion reactions
Sickle cell disease related emergencies
Acute chest syndrome
Hyperhemolysis syndrome
Systemic Inflammatory and Thrombotic Emergencies
Hemophagocytic Lymphohistiocytosis
Catastrophic Antiphospholipid Syndrome
Malignancy-related hematological emergencies
Hyperleukocytosis and leukostasis
Acute promyelocytic leukemia
Tumor lysis syndrome
Febrile neutropenia
Cytokine release syndrome

The International Society on Thrombosis and Hemostasis scoring system provides a standardized approach to diagnosis, incorporating platelet count, fibrin-related markers, prolonged coagulation times, and fibrinogen levels.[7] Routine laboratory abnormalities in DIC [Table 2] include low platelet count, prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), increased fibrin degradation products levels, and decreased levels of protease inhibitors such as protein C, protein S, and antithrombin.[8]

Management of DIC focuses primarily on treating the underlying condition while providing supportive care. Platelet transfusion is recommended for counts below 10,000–20,000/μL in bleeding patients or below 50,000/μL in those requiring invasive procedures. Fresh frozen plasma, typically at 15 mL/kg–30 mL/kg, and cryoprecipitate may be considered for patients with prolonged coagulation times and active bleeding. Packed red cells may be given to maintain the hemoglobin >8 g/dL. Anticoagulation with heparin may be required in cases where significant thrombosis is present, as it can inhibit additional clotting cascade activation. DIC patients without active hemorrhage should be given preventive anticoagulation using heparin or low-molecular-weight heparin preparations.[8-11] The use of antifibrinolytic agents remains controversial and should be avoided in cases with predominant thrombotic features.[11]

Trauma-induced coagulopathy (TIC)

TIC can be defined as a condition of endogenous hypercoagulation observed in the immediate post-traumatic period, that is, within 1 h of trauma. It represents a distinct entity from DIC, characterized by early coagulopathy.[12] TIC is present in approximately one-third of patients who report major trauma. This condition involves endothelial activation, protein C pathway activation, and hyperfibrinolysis.[12,13]

Recognition requires understanding of the mechanism of injury and rapid assessment of coagulation parameters using standard coagulation test and functional viscoelastic assays.[12,14] Traditional tests (PT, aPTT, fibrinogen, and platelets) do not reflect clinical phenotypes well and require a long time for analysis, whereas viscoelastic tests such as thromboelastography and thromboelastometry provide faster, more comprehensive coagulation assessment, identifying TIC stages (hyperfibrinolysis, hypocoagulation,

Table 2: Laboratory values variation in various hematological emergencies.

Condition	Platelets ($\times 10^3/\mu L$) Normal: 150–400	PT/INR Normal: 10–13s INR 0.8–1.2	aPTT (s) Normal: 25–35	Fibri-nogen (mg/dL) Normal: 200–400	LDH (U/L) Normal: 140–280	Hapto-globin (mg/dL) Normal: 40–200	Ferritin (ng/mL) Normal: 15–150	Schistocytes	D-Dimer (mg/L) Normal: <0.5
DIC (Acute)	<100 (moderate) <50 (severe)	↑ (50%)	↑ (50%)	↓ to Normal	Mild ↑	Variable	↑	Present (Few)	↑
TTP	<30	Normal	Normal	Normal	↑	↓	Normal	Abundant	Normal to Mild ↑
aHUS	<100 ↓	Normal	Normal	Normal	↑	↓	Normal	Present	Mild ↑
AIHA	Normal	Normal	Normal	Normal	↑ ↑	↓	Normal	Present (Few)	Normal
Acute Transfusion Reaction	Normal	Normal	Normal	Normal	↑ ↑	↓ ↓	Normal	Absent or Few	Normal
HLH	↓	Mild ↑	Mild ↑	↓	Mild ↑	Variable	↑	Absent	Mild ↑
Hyper-leukocytosis	Variable	Mild ↑	Mild ↑	Mild ↓	Mild ↑	Normal	↑	Absent	Mild ↑
ACS (Sickle Cell)	Normal	Normal	Normal	Normal	Mild ↑	Mild ↓	Normal	Present	Normal

Normal/Expected, Mildly abnormal, Severely abnormal, Variable. DIC: Disseminated intravascular coagulation, TTP: Thrombotic thrombocytopenic purpura, aHUS: Atypical hemolytic uremic syndrome, AIHA: Autoimmune hemolytic anemia, HLH: Hemophagocytic lymphohistiocytosis, ACS: Acute chest syndrome, PT: Prothrombin time; INR: International normalized ratio, aPTT: Activated partial thromboplastin time, LDH: Lactate dehydrogenase

and hypercoagulation), and guiding targeted treatment while preventing unnecessary transfusions.[12,14] TIC is related to worse outcomes, among which are increased rates of transfusion, infection, thromboembolism, acute lung injury, multi-organ failure, and death.[12]

The management approach emphasizes damage control resuscitation with balanced transfusion ratios, typically 1:1:1 for red blood cells (RBCs), fresh frozen plasma, and platelets. Early administration of tranexamic acid within 3 h of injury has been shown to reduce mortality in bleeding trauma patients. Massive transfusion protocols should be activated early in cases of significant hemorrhage.[12,15,16]

Thrombotic emergencies

Thrombotic thrombocytopenic purpura (TTP)

TTP represents a hematological emergency with high mortality if untreated. TTP is characterized by thrombocytopenia, microangiopathic hemolytic anemia, neurological symptoms, fever, and renal dysfunction.[17] The underlying pathophysiology involves severe deficiency of A Disintegrin and Metalloproteinase with a Thrombospondin type 1 motif, member 13 (ADAMTS13), a metalloprotease that cleaves von Willebrand factor multimers.

Early recognition is crucial as delays in treatment significantly increase mortality. Symptoms are often non-specific initially (weakness, confusion, and nausea), progressing to organ-specific manifestations. Neurological complications affect ~10% of patients, while renal failure occurs in over half of cases. Cardiac involvement may present with chest pain and elevated troponin.[12,18,19] Routine laboratory abnormalities include severe thrombocytopenia (platelet count usually <30000/μL), decreased hemoglobin and hematocrit, significantly elevated lactate dehydrogenase (LDH), low haptoglobin, increased indirect bilirubin, and presence of schistocytes on peripheral blood smear. The PLASMIC score has been validated as a useful tool for rapid assessment of TTP probability, incorporating platelet count, hemolysis markers, absence of active cancer, absence of stem cell transplantation, creatinine level, INR, and mean corpuscular volume.[20] Although plasma ADAMTS13 activity (<10 IU/dL) is highly specific for TTP, a high PLASMIC score should prompt immediate initiation of therapeutic plasma exchange even before ADAMTS13 results are available.[17,21]

Management centers on immediate therapeutic plasma exchange, which should be initiated within hours of suspected diagnosis.[22] Daily plasma exchange is continued until platelet normalization and resolution of hemolysis markers. Corticosteroids are typically used as adjunctive therapy. Rituximab may be considered for refractory cases or those with severely deficient ADAMTS13 activity. Platelet transfusion is contraindicated unless life-threatening bleeding occurs, as it may worsen thrombotic complications.[17]

Atypical hemolytic uremic syndrome (aHUS)

aHUS presents with the triad of thrombocytopenia, microangiopathic hemolytic anemia, and acute kidney injury, but unlike typical hemolytic uremic syndrome, it is not associated with Shiga toxin.[23] The condition results from dysregulation of the alternative complement pathway due to genetic mutations or acquired inhibitors.[23]

Recognition requires differentiation from TTP and other thrombotic microangiopathies. The absence of severe ADAMTS13 deficiency (<10% activity) and predominant renal involvement help distinguish aHUS from TTP.[24] Complement studies, including C3, C4, and CH50 levels, along with genetic testing for complement regulatory proteins, aid in diagnosis.[23,24]

Treatment involves complement inhibition with eculizumab, which has dramatically improved outcomes in aHUS. Early initiation of eculizumab is crucial, as delays may result in irreversible organ damage. Plasma exchange may provide temporary benefit but is not as effective as in TTP. Supportive care includes management of acute kidney injury and hypertension.[23,24]

Hemolytic emergencies

Autoimmune hemolytic anemia (AIHA)

AIHA can present as a hematological emergency when hemolysis is severe and rapid. AIHA is characterized by antibody-mediated destruction of RBCs, leading to anemia, hemoglobinemia, and potentially life-threatening complications.[25]

Warm AIHA

Warm AIHA accounts for 70–80% of cases and involves immunoglobulin G antibodies that bind RBCs at body temperature, targeting Rh antigens and causing splenic extravascular hemolysis. It may be idiopathic or secondary to lymphoproliferative disorders, autoimmune diseases, infections, or medications.[25,26] Patients present with acute anemia, jaundice, dark urine, fatigue, dyspnea, chest pain, tachycardia, and in severe cases, high-output heart failure and shows splenomegaly on physical examination. Laboratory findings include reticulocytosis, elevated LDH, decreased haptoglobin, elevated indirect bilirubin, and positive direct antiglobulin test (DAT).[25,26]

First-line treatment is prednisolone at 1–1.5 mg/kg daily, with 70–85% showing initial improvement within 1–2 weeks. Transfusion support may be necessary despite challenging cross-matching. Second-line therapies include rituximab, splenectomy, or immunosuppressive agents such as azathioprine, cyclosporine, or mycophenolate mofetil for refractory cases.[25-27]

Cold AIHA

Cold AIHA represents 15–25% of cases, mediated by immunoglobulin M cold agglutinins that bind RBCs below body temperature, targeting the I/i antigen system. It may be primary, occurring as cold agglutinin disease, or secondary to infections such as Mycoplasma pneumoniae and Epstein-Barr virus, or lymphoproliferative disorders. Hemolysis occurs through complement activation (intravascular) and hepatic removal (extravascular). Patients experience cold-triggered symptoms including acrocyanosis, Raynaud's phenomenon, and livedo reticularis. Laboratory tests show positive DAT with anti-C3d positivity and elevated cold agglutinin titers.[25,26]

Management differs significantly: Corticosteroids are ineffective. Treatment focuses on cold avoidance, rituximab therapy, treating underlying conditions, and emerging complement inhibitors such as eculizumab or sutimlimab. Transfusions require blood warmers, and splenectomy is generally ineffective since hemolysis occurs primarily in the liver.[25-27]

Hemolytic transfusion reactions

Acute hemolytic transfusion reactions represent preventable but potentially fatal complications of blood transfusion. These reactions typically result from ABO incompatibility and can lead to DIC, acute kidney injury, and cardiovascular collapse.[28]

Recognition requires immediate suspicion when patients develop fever, chills, back/chest pain, agitation, burning at infusion site, headache, nausea, or dyspnea during or shortly after transfusion. Objective signs include fever, skin changes, tachycardia, hypotension, and reddish urine. In anesthetized patients lacking subjective symptoms, shock, hemoglobinuria, and hemorrhage may be initial manifestations. Laboratory findings include hemoglobinemia, decreased haptoglobin, elevated LDH levels, and positive DAT.[28] The transfusion must be stopped immediately, and blood samples from both patient and donor unit should be sent for compatibility testing.[28,29]

Management focuses on supportive care including aggressive fluid resuscitation to maintain renal perfusion, monitoring for DIC, and treatment of shock if present. Corticosteroids and antihistaminics are administered to relieve the symptoms. In the case of a severe transfusion reaction, basic life support has to start at once. Prevention remains the most important aspect, emphasizing proper patient identification and blood product verification procedures.[28]

Sickle cell disease-related emergencies

Acute chest syndrome (ACS)

ACS represents the leading cause of death in adults with sickle cell disease and a frequent indication for ICU admission.[30] ACS is defined as a new pulmonary infiltrate,

involving at least one complete lung segment, accompanied by fever, chest pain, dyspnea, wheezing, or cough in patients with sickle cell disease. The pathophysiology involves vaso-occlusion in pulmonary vessels, fat embolism from bone marrow necrosis, and infection.[30-32]

Recognition requires maintaining high clinical suspicion in sickle cell patients presenting with respiratory symptoms. Clinical presentation may include chest pain, fever, cough, tachypnea or wheezing. Pain in the abdomen, limb, rib, or sternum is also frequent, along with intercostal retractions, the use of accessory muscles for respiration, and hypoxemia. Chest radiography reveals new infiltrates, though these may lag behind clinical symptoms. Patient can also present with multiorgan failure syndrome due to fat and marrow embolization. Laboratory findings often show decreased hemoglobin from baseline, elevated LDH, and leukocytosis.[30]

Management involves aggressive supportive care including oxygen supplementation, fluid therapy, adequate analgesia with parenteral opioids (avoiding oversedation), incentive spirometry, and bronchodilators if indicated. Simple transfusion is recommended for hemoglobin levels below 9 g/dL, while exchange transfusion may be necessary for severe cases with multiorgan involvement. Broad-spectrum antibiotics should cover typical and atypical organisms. Corticosteroids may be beneficial but require careful consideration due to potential rebound effects. Some patients require mechanical ventilation, especially those having occult pulmonary hypertension and cor pulmonale.[30,31,33]

Hyperhemolysis syndrome

Hyperhemolysis syndrome is a rare but potentially fatal complication in sickle cell patients, characterized by acute hemolysis with hemoglobin levels falling below pre-transfusion values despite transfusion therapy.[34] This condition involves both alloantibody and autoantibody-mediated destruction of transfused and autologous RBCs. The possible mechanisms include bystander hemolysis, suppression of erythropoiesis, and destruction of RBCs by activated macrophages.[34,35]

Recognition requires awareness of the paradoxical drop in hemoglobin following transfusion in sickle cell patients. Patients may present with severe anemia, fever, jaundice, back/abdominal/joint pain, fatigue, and dark urine. Laboratory findings may include unexpected reticulocytopenia, elevated LDH, indirect hyperbilirubinemia, and raised C-reactive protein levels. Hyperhaemolysis syndrome has acute (within 7 days, negative DAT, no new antibodies) and delayed (after 7 days, positive DAT with antibody formation) variants.[34-37]

Management focuses on discontinuing transfusions and providing supportive care. Immunosuppressive therapy with corticosteroids and intravenous immunoglobulin (IVIG) may be beneficial. Rituximab and eculizumab have shown good response. Erythropoietin can stimulate endogenous RBC production. Exchange transfusion should be avoided as it may worsen hemolysis. Close monitoring for complications including acute kidney injury and cardiovascular collapse is essential.[36-39]

Systemic inflammatory and thrombotic emergencies

Hemophagocytic lymphohistiocytosis (HLH)

HLH is a life-threatening condition characterized by excessive immune activation and inflammatory response, often triggered by infections, malignancies, or autoimmune disorders.[40] HLH can be primary (genetic) or secondary (acquired), with secondary HLH being more common in adult ICU patients.[41]

Recognition relies on the HLH-2004 criteria, which include fever, splenomegaly, bicytopenia, hypertriglyceridemia ± hypofibrinogenemia, elevated ferritin (>500 ng/mL), low or absent NK-cell activity, elevated interleukin-2 receptor subunit alpha (sIL2Ra) levels (≥2400 U/mL), and hemophagocytosis in bone marrow. Diagnosis is established by the presence of at least 5 of the combined 8 total criteria.[41,42] The HScore calculator provides a probability-based approach to diagnosis, incorporating clinical and laboratory parameters. Ferritin levels above 10,000 ng/mL are highly suggestive of HLH.[43]

Management requires prompt initiation of immunosuppressive therapy, after excluding serious infections, to control the hyperinflammatory response. The HLH-2004 protocol includes treatment with etoposide, dexamethasone, and cyclosporine A as first-line therapy, with intrathecal methotrexate and steroid administration reserved for specific cases. Following initial management, hematopoietic stem cell transplantation is indicated for individuals with hereditary forms of the condition or confirmed genetic mutations, as well as those experiencing severe, ongoing, or recurrent disease manifestations. Emerging therapies include emapalumab (anti-Interferon-gamma antibody) and ruxolitinib, which showed promise in murine models by improving survival and reducing inflammatory markers.[41,44,45] Treatment of underlying triggers is crucial, particularly in infection-associated HLH. Supportive care includes management of cytopenias, coagulopathy, and organ dysfunction. Early recognition and treatment are critical, as untreated HLH has a mortality rate approaching 100%.[40]

Catastrophic antiphospholipid syndrome (CAPS)

CAPS represents the most severe form of antiphospholipid syndrome, characterized by widespread thrombotic

microangiopathy affecting multiple organs simultaneously.[46] The underlying disease mechanism centers on antiphospholipid antibodies, especially anti-beta-2-glycoprotein I (aβ2GPI), which trigger endothelial cell activation, cytokine release, and a prothrombotic state.[47] CAPS accounts for <1% of all antiphospholipid syndrome cases but carries a mortality rate of approximately 30–50%.[48]

Recognition requires identifying acute multiorgan thrombotic events in patients with antiphospholipid antibodies. Clinical presentation typically involves thrombosis in at least three organ systems over days to weeks, commonly affecting kidneys, lungs, brain, heart, gastrointestinal tract, and skin.[48] Renal involvement occurs with varying kidney dysfunction, while pulmonary complications can include acute respiratory distress syndrome and pulmonary embolism. Neurological manifestations can present as stroke or encephalopathy. Cardiac involvement affects half of patients, primarily through myocardial infarction or valve disease, with Libman-Sacks endocarditis occurring in some patients, especially in Systemic lupus erythematosus (SLE)-associated CAPS. Patients frequently exhibit cytokine storm features and microangiopathic phenomena, including livedo and Raynaud's syndrome. During pregnancy, severe HELLP syndrome represents a major CAPS manifestation, while purpura occurs less commonly.[48] Laboratory findings include positive antiphospholipid antibodies (anticardiolipin, aβ2GPI, or lupus anticoagulant) and evidence of microangiopathic hemolytic anemia.[46-48]

Management involves triple therapy consisting of anticoagulation, corticosteroids, and plasma exchange or IVIG.[48,49] Immediate anticoagulation with heparin is essential, though the optimal intensity remains debated. High-dose corticosteroids (0.5–1 mg/kg, followed by tapering) help control the inflammatory response. Plasma exchange removes circulating antibodies and inflammatory mediators, while IVIG provides immunomodulation. In patients with SLE, cyclophosphamide should be considered. In refractory or relapsing cases, new therapies, such as rituximab and possibly eculizumab, may be options, but need further study. Treatment of precipitating factors such as infections or surgery is crucial.[48,49]

Malignancy-related hematological emergencies

Hyperleukocytosis and leukostasis

Hyperleukocytosis, defined as white blood cell (WBC) count exceeding 100,000/μL, can lead to leukostasis with potentially fatal complications including pulmonary and cerebral dysfunction.[50] This emergency is most commonly associated with acute leukemias, particularly acute myeloid leukemia and acute lymphoblastic leukemia.[50,51]

Leukostasis refers to clinical symptoms and complications caused by hyperleukocytosis.[51] Clinical diagnosis is challenging as symptoms mimic infections or hemorrhagic complications. The Novotny score aids diagnosis, with scores ≥3 indicating high probability.[51,52] Approximately 44–50% of acute myeloid leukemia (AML) patients with WBC >100,000/μL develop leukostasis, though it can occur at lower counts. Primary organ involvement includes lungs (dyspnea, hypoxemia, and respiratory failure), brain (confusion, headache, and focal deficits), and kidneys. Imaging may show bilateral pulmonary infiltrates and intracranial hemorrhage. Additional manifestations include visual impairment, tinnitus, myocardial ischemia, limb ischemia, and priapism. Hemorrhagic and thromboembolic complications frequently accompany tissue damage from leukocyte stasis and infiltration.[51]

Management requires immediate cytoreduction through leukapheresis when available, along with urgent initiation of chemotherapy. Hydroxyurea may be used as a temporizing measure. Supportive care includes aggressive hydration, allopurinol for tumor lysis syndrome (TLS) prevention, and management of electrolyte abnormalities. Platelet transfusion should be avoided unless severe bleeding occurs, as it may worsen hyperviscosity. RBC transfusions require caution due to their viscosity-increasing effects – they should be administered only when hemoglobin drops below 7–8 g/dL in stable patients, avoiding levels exceeding 10 g/dL post-transfusion. Coagulation defects need correction.[50,53]

Acute promyelocytic leukemia (APML)

APML (AML-M3) represents a unique hematological emergency characterized by life-threatening coagulopathy and high early mortality if untreated. Accounting for 10–15% of AML cases, APML features the characteristic t(15;17) translocation creating the promyelocytic leukemia-retinoic acid receptor alpha. (PML-RARA) fusion gene, causing differentiation block at the promyelocyte stage.[54]

Up to 90% of patients present with severe coagulopathy due to abnormal promyelocytes releasing procoagulant substances, causing DIC and hyperfibrinolysis.[54,55] Hemorrhagic complications, particularly intracranial hemorrhage, occur in 10-30% during the first week. Patients present with fatigue, fever, infections, and bleeding manifestations including petechiae, ecchymoses, epistaxis, and severe gastrointestinal or pulmonary hemorrhage.[54]

Laboratory findings include pancytopenia, abnormal promyelocytes with Auer rods, prolonged coagulation times, severe hypofibrinogenemia (<100 mg/dL), elevated D-dimer, and thrombocytopenia (<40,000/μL).[54,55] Treatment should begin on morphological suspicion without awaiting molecular confirmation.

Aggressive coagulopathy management is critical: maintain fibrinogen >150 mg/dL with cryoprecipitate and platelets >30,000–50,000/μL. All-trans retinoic acid (ATRA)

at 45 mg/m^2/day should start immediately, inducing promyelocyte differentiation and reducing coagulopathy within 2–5 days. For low-to-intermediate risk patients (WBC <10,000/μL), ATRA plus arsenic trioxide (0.15 mg/kg/day) without chemotherapy is standard. High-risk patients (WBC >10,000/μL) require additional chemotherapy.[54,55]

Differentiation syndrome occurs in 15–30% of patients within 2–3 weeks, presenting with fever, respiratory distress, pulmonary infiltrates, weight gain, and hypotension. Immediate treatment with dexamethasone 10 mg intravenous (IV) every 12 h is essential.[54,55] Other complications include QTc prolongation requiring electrolyte monitoring and hepatotoxicity. Modern ATRA-arsenic trioxide therapy achieves complete remission rates exceeding 95% and cure rates of 90–95%, transforming APML from highly fatal to one of the most curable leukemias.[54,55]

TLS

TLS is an oncologic emergency resulting from rapid malignant cell breakdown, releasing intracellular contents and causing severe metabolic derangements. TLS occurs spontaneously or following chemotherapy initiation, with acute kidney injury developing in 25–50% of high-risk cases.[56,57]

Key metabolic consequences include hyperkalemia causing potentially fatal cardiac arrhythmias, hyperphosphatemia, leading to calcium-phosphate precipitation in renal tubules, hypocalcemia causing neuromuscular irritability and seizures, and hyperuricemia from nucleic acid breakdown causing uric acid nephropathy.[56-58]

High-risk malignancies include Burkitt lymphoma, high-grade non-Hodgkin lymphomas, and acute leukemias with high WBC counts. Patient factors include renal dysfunction, elevated baseline uric acid, markedly elevated LDH, and bulky disease.[56,57]

Prevention is paramount. Aggressive IV hydration maintains urine output >100–150 mL/h using 2–3 L/m^2/day of isotonic fluids, avoiding potassium and calcium. Allopurinol (300–600 mg/day) prevents new uric acid formation, while rasburicase (0.15–0.2 mg/kg IV) rapidly converts existing uric acid to soluble allantoin within 4 hours. Treatment of established TLS requires continuous cardiac monitoring, hyperkalemia management with calcium gluconate and insulin-dextrose, phosphate binders avoiding calcium, treating only symptomatic hypocalcemia, and rasburicase for hyperuricemia. Hemodialysis is indicated for severe refractory electrolyte abnormalities. Early recognition and aggressive prophylaxis are crucial for improving outcomes.[56-58]

Febrile neutropenia

Febrile neutropenia represents a medical emergency in cancer patients, with the potential for rapid progression to septic shock and death. According to the European Society for Medical Oncology guidelines, febrile neutropenia is defined as fever consisting of either a single oral temperature of ≥38.3°C (101°F) or a temperature ≥38.0°C (100.4°F) sustained over 1 h, accompanied by neutropenia defined as an absolute neutrophil count <500 cells/μL or an absolute neutrophil count <1000 cells/μL with predicted decline to <500 cells/μL over the next 48 h.[59] Severe neutropenia is defined as counts under 500 cells/μL, while profound neutropenia involves counts below 100 cells/μL.[59,60]

Recognition requires a high index of suspicion in neutropenic patients presenting with fever, even in the absence of localizing signs of infection. The usual inflammatory response may be absent due to neutropenia, making clinical assessment challenging. Pain and tenderness may be the only indicators of infection. Blood cultures should be obtained from all lumens of central venous catheters and peripheral sites.[60]

Management involves immediate initiation of broad-spectrum antibiotics within one hour of presentation. Monotherapy with antipseudomonal beta-lactam agents such as cefepime, carbapenems, or piperacillin/tazobactam has been recommended.[61] Empirical antibiotic selection should cover gram-positive and Gram-negative bacteria, with consideration of local resistance patterns and patient risk factors. Antifungal therapy may be added for patients with persistent fever despite antibacterial treatment. Granulocyte colony-stimulating factor may be considered in high-risk patients.[60]

Cytokine release syndrome (CRS)

CRS has emerged as a significant hematological emergency, particularly in the era of immunotherapy and chimeric antigen receptor T-cel (CAR-T) cell therapy.[62] CRS involves excessive release of inflammatory cytokines, leading to systemic inflammation and potentially life-threatening complications.

Recognition involves identifying fever, hypotension, capillary leak syndrome, coagulopathy with widespread clotting dysfunction, and organ dysfunction in patients receiving immunotherapy or following stem cell transplantation. Severity grading systems help guide treatment decisions, with severe CRS characterized by hypotension requiring vasopressors and hypoxemia requiring mechanical ventilation. Common laboratory findings in CRS patients encompass cytopenias, elevated serum creatinine, hepatic enzyme elevations, abnormal clotting studies, and markedly increased C-reactive protein levels reflecting the intense inflammatory state.[62]

Management includes supportive care and treatment with antihistamines, antipyretics and fluids for mild cases, and

cytokine blockade with tocilizumab (anti-IL-6 receptor antibody) for moderate to severe cases. Corticosteroids may be used for severe cases refractory to tocilizumab. Early recognition and grading are crucial for optimal outcomes.[62,63]

MULTIDISCIPLINARY CARE APPROACHES

A systematic algorithmic approach to hematological emergencies facilitates rapid recognition and appropriate management [Figure 1]. This standardized workflow ensures comprehensive evaluation while minimizing delays in critical interventions.

Team composition and coordination

Effective management of hematological emergencies requires coordinated multidisciplinary care involving multiple specialties. The core team typically includes intensivists, hematologists, transfusion medicine specialists, and specialized nursing staff. Additional consultants may include nephrology, neurology, infectious diseases, and pharmacy specialists depending on the specific condition.[2]

Communication and coordination are essential for optimal outcomes. Regular multidisciplinary rounds, clear communication protocols, and shared decision-making processes improve care quality and patient safety. Electronic health records with real-time laboratory integration facilitate rapid decision-making and treatment adjustments.

Transfusion medicine integration

Transfusion medicine plays a crucial role in managing hematological emergencies. Close collaboration between ICU teams and blood bank personnel ensures appropriate blood product selection, compatibility testing, and monitoring for transfusion reactions. Massive transfusion protocols require predefined triggers, blood product ratios, and monitoring parameters.

Special considerations include management of patients with rare blood types, multiple alloantibodies, or religious objections to transfusion. Alternatives to transfusion, such as iron supplementation, erythropoietin, and blood conservation techniques, should be considered when appropriate.[64]

Procedural interventions

Many hematological emergencies require urgent procedural interventions such as therapeutic plasma exchange, leukapheresis, or bone marrow biopsy. These procedures require coordination between ICU staff, apheresis services, and interventional teams. Patient stability assessment, vascular access planning, and post-procedure monitoring protocols are essential components of care.

QUALITY IMPROVEMENTS AND OUTCOMES

Standardized protocols

Implementation of standardized protocols for recognition and management of hematological emergencies improves outcomes and reduces variability in care. These protocols should include diagnostic criteria, treatment algorithms, and monitoring parameters. Regular protocol review and updates based on emerging evidence ensure continued relevance and effectiveness.[1]

Performance metrics

Key performance indicators for hematological emergencies include time to diagnosis, time to treatment initiation, transfusion ratios, and clinical outcomes such as mortality and length of stay. Regular monitoring of these metrics identifies areas for improvement and tracks progress over time.

Education and training

Ongoing education for ICU staff regarding recognition and initial management of hematological emergencies is essential. Simulation-based training, case-based discussions, and multidisciplinary educational conferences enhance knowledge and skills. Regular competency assessments ensure maintenance of proficiency.[65]

FUTURE DIRECTIONS AND EMERGING THERAPIES

Novel therapeutic approaches

Emerging therapies for hematological emergencies include complement inhibitors for thrombotic microangiopathies, novel anticoagulants for bleeding disorders, and targeted therapies for malignancy-related complications. Gene therapy approaches for inherited bleeding disorders and hemoglobinopathies show promise for future applications.

Precision medicine

Advances in genetic testing and biomarker identification enable more personalized approaches to treatment. Pharmacogenomics may guide drug selection and dosing, while genetic testing can identify patients at risk for specific complications.

Technology integration

Point-of-care testing devices, artificial intelligence-assisted diagnosis, and telemedicine consultation expand capabilities for rapid diagnosis and treatment. Electronic decision support systems can help guide treatment selection and monitor for potential complications.

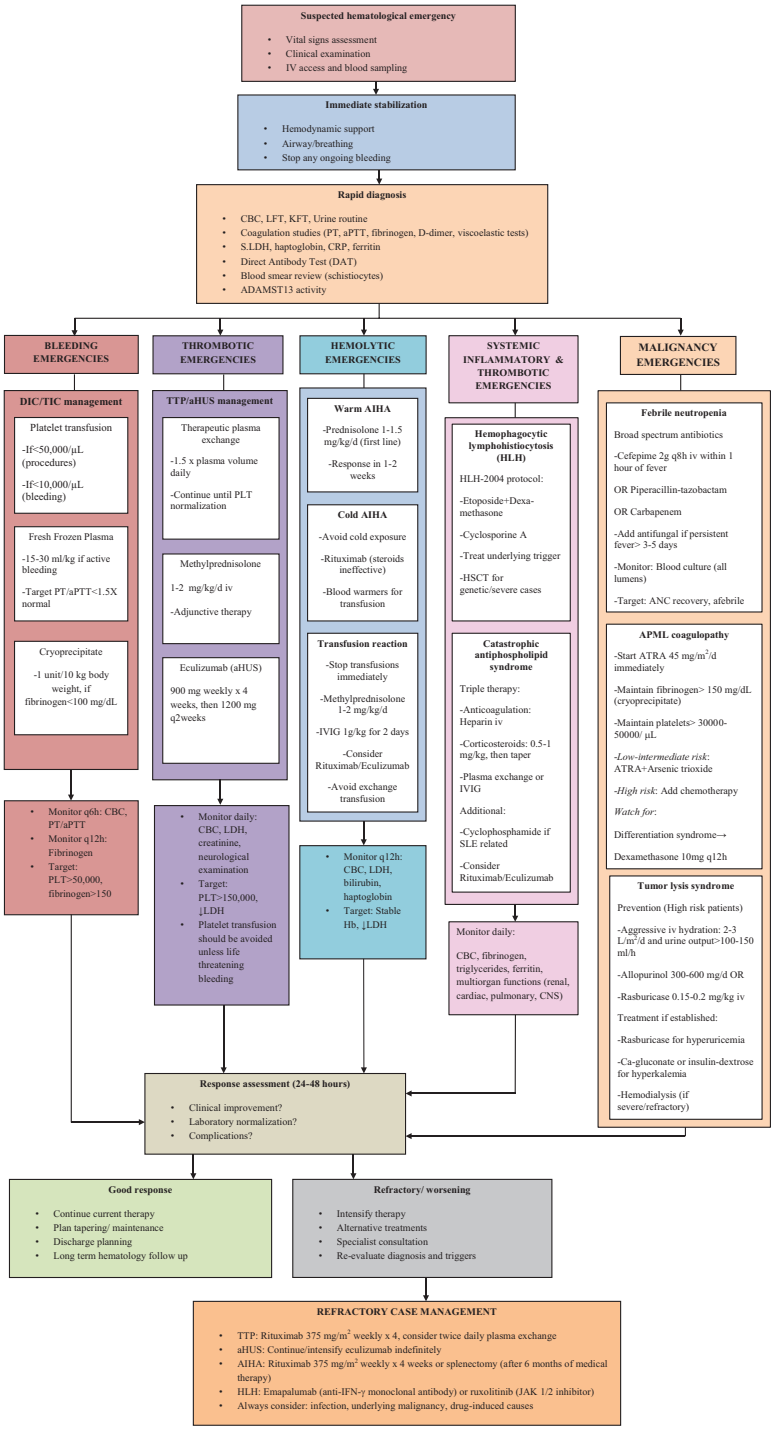

Figure 1: Algorithmic approach to recognition and management of hematological emergencies in intensive care unit ADAMTS13: A Disintegrin and Metalloproteinase with a ThromboSpondin type 1 motif, member 13, ANC: Absolute neutrophil count, aPTT: Activated partial thromboplastin time, CBC: Complete blood count, CRP: C-reactive protein, DAT: Direct antiglobulin test, INR: International normalized ratio, KFT: Kidney function tests, LDH: Lactate dehydrogenase, LFT: Liver function tests, PLT: Platelets, PT: Prothrombin time, S.LDH: Serum lactate dehydrogenase, aHUS: Atypical hemolytic uremic syndrome, AIHA: Autoimmune hemolytic anemia, APML: Acute promyelocytic leukemia, ATRA: All-trans retinoic acid, CNS: Central nervous system, DIC: Disseminated intravascular coagulation, TIC: Trauma induced coagulopathy, HLH: Hemophagocytic lymphohistiocytosis, HSCT: Hematopoietic stem cell transplantation, IFN-γ: Interferon-gamma, IV: Intravenous, IVIG: Intravenous immunoglobulin, JAK: Janus kinase, SLE: Systemic lupus erythematosus, TTP: Thrombotic thrombocytopenic purpura.

CONCLUSION

Hematological emergencies in ICUs represent complex, time-sensitive conditions requiring immediate recognition and coordinated multidisciplinary intervention. Success in managing these critical situations hinges on maintaining clinical vigilance, implementing evidence-based diagnostic protocols, and ensuring seamless collaboration among specialized teams. The diverse spectrum of conditions – from coagulopathies and thrombotic microangiopathies to hemolytic crises and malignancy-related complications – demands specialized expertise and standardized management approaches. Early intervention remains the cornerstone of improved patient outcomes, with delays in recognition or treatment significantly increasing morbidity and mortality. As therapeutic advances continue to emerge, including precision medicine approaches and novel targeted therapies, healthcare systems must prioritize ongoing education, protocol standardization, and quality improvement initiatives. The future success of hematological emergency management will depend on integrating technological innovations with fundamental principles of rapid diagnosis, appropriate treatment, and coordinated multidisciplinary care delivery.

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